Hyperthyroidism Masked by Psychotic Depression

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A HYPERMETABOLIC STATE and manifestations resembling those of overactivity of the autonomic nervous system are characteristic of hyperthyroidism.[15,19,23] These features of the disease are usually reflected in the patient's behavior by nervousness, irritability, emotional lability, and even at times by hypomania.[7,9,15,23] Infrequently, however, the characteristic signs and symptoms are not present. In such cases, the condition is referred to as non-active, apathetic or masked hyperthyroidism.*

Recently, we had occasion to study a patient with masked hyperthyroidism. The case was unusual in that a psychotic depression occurred during the course of illness and may have obscured some of the usual stigmata of the hyperthyroid state. Manifestations of thyrotoxicosis appeared only after a series of electroshock treatments and resolution of the depressive state.

REPORT OF A CASE

A 39-year-old Caucasian housewife, mother of three children, was referred to the Langley Porter Neuropsychiatric Institute on July 25, 1961, because of depression of five months' duration. A transient episode of depression had occurred once previously when the patient was 22 years old. She had felt well thereafter until the summer of 1960, when she first noticed difficulty in swallowing and found that her legs tired easily when climbing stairs. During the next six months she gradually became nervous, her appetite decreased and she lost 23 pounds in weight. At the time of a routine examination by a physician in December, the pulse rate was 120 per minute and regular, and the blood pressure was 120/80 mm of mercury. Her skin was warm and dry, and she had a fine tremor of the hands. The thyroid was enlarged, but no bruit or palpable nodules were present. The protein-bound iodine level was 25 μgm per 100 ml (repeated), and radioactive iodine uptake by the thyroid was 97 per cent in 24 hours. An electrocardiogram showed sinus tachycardia. No abnormalities were seen in a roentgenogram of the chest. A diagnosis of hyperthyroidism was made, and methimazole, 5 mg, and phenobarbital, 15 mg, both to be given three times a day, were prescribed.

By the middle of February 1961 the patient's pulse rate had decreased to 80 per minute. She tired easily and was unable to maintain her meticulous standards of housekeeping or take her accustomed part in church activities. Gradually she became anxious, suspicious and depressed. Therapy with radioactive iodine was planned, and the antithyroid medication was discontinued. In May the planned treatment was cancelled because of the patient's extreme tension and her fear of a radioactive agent. Her depression increased, and after the death of a close friend she began to feel worthless and guilt-ridden and to have ideas of reference. When she was next examined by her physician in June, a few weeks after discontinuing methimazole, she appeared extremely depressed. The pulse rate was 120 per minute, and blood pressure 110/80 mm of mercury. The fine tremor of the hands was still present, and the thyroid enlargement was greater than in December. The protein-bound iodine level was 4.5 μgm per 100 ml, thyroidal radioactive iodine uptake 99 per cent at 24 hours and basal metabolic rate minus 11. On the basis of these findings, methimazole, 5 mg twice daily, was again prescribed. At her next visit approximately one month later, the thyroid gland had decreased in size, the pulse was 120, and the hand tremor had disappeared. Psychological symptoms, however, had increased to such a degree that psychiatric evaluation was considered advisable. The patient was then referred to the Acute Treatment Service of the Langley Porter Neuropsychiatric Institute.

The course of illness, correlated with the results of thyroid function studies and treatment, is shown in Figure 1.

Relevant facts in the family and past histories were as follows. The patient, the only child of middle-class parents, had been born in a small Midwestern community. Both parents had a history of thyroidectomy because of goiter; neither had experienced psychiatric difficulties at any time. The patient developed into a religious, meticulous, hard-driving person. At the age of 22, while taking postgraduate courses in another town, working to supplement her income, and directing a church youth group, she had "influenza." Because the illness persisted, she was unable to carry out her commitments, and became depressed and morose. She had six interviews with a psychologist, but because the depression continued she left school and returned to her home town. The depression subsided gradually. In her late 20's she married; she had been a competent housewife and mother until the onset of the present illness.

At the time of admission, the patient was a slen-

*Reference Nos. 1, 5, 6, 8, 11, 13, 14, 17, 22, 24, 25.

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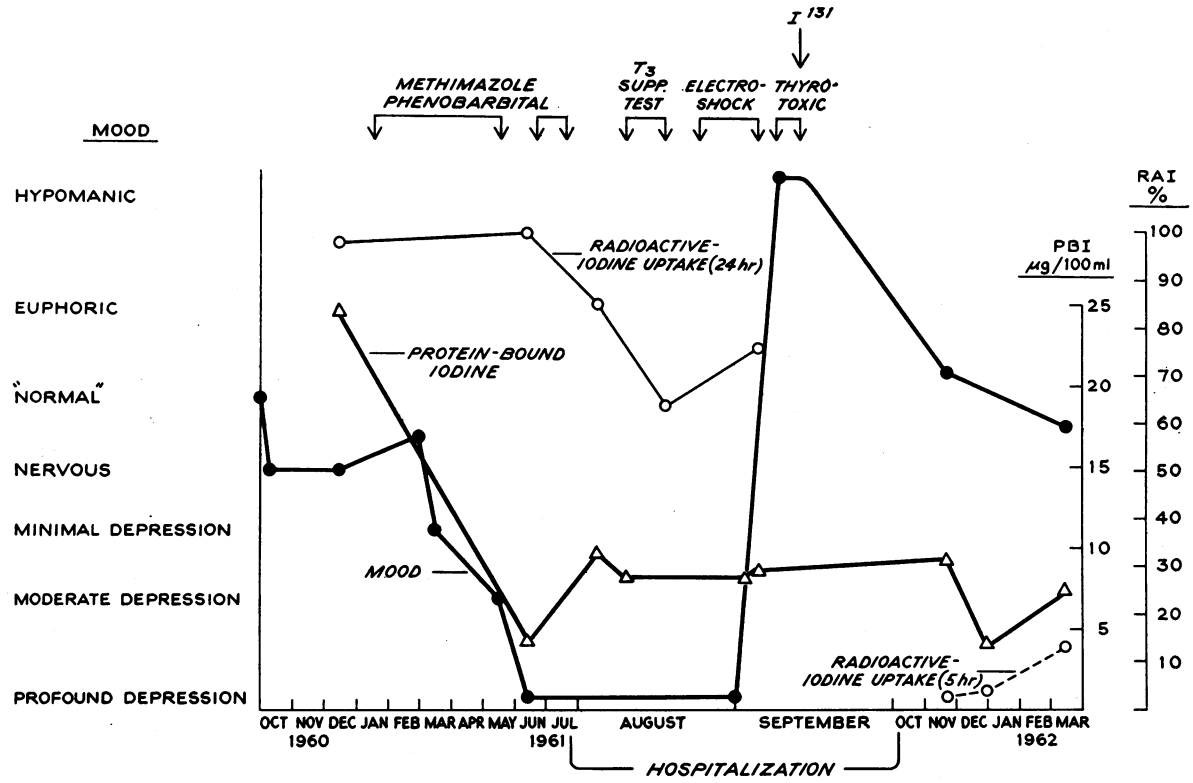

Figure 1.—Mood and thyroid function in patient with hyperthyroid state masked by psychotic depressive state. After electroshock treatments, depression cleared and clinical signs of thyrotoxicosis appeared.

der woman, 66 inches tall, weighing 120 pounds. She was sloppily dressed and slow moving, and appeared older than her stated age. Blood pressure was 118/70 mm of mercury, and pulse rate 108 per minute. Her skin was warm but not moist, and pigmentation was moderately increased. Her hair was of normal texture. The thyroid gland was diffusely enlarged; no nodules were palpable and no bruit was heard. Ocular, cardiopulmonary and neurologic examinations showed no abnormalities. No tremor of the hands was present. During the psychiatric interviews, the patient was indecisive and suspicious, and appeared excessively concerned about her children's health as well as her own. Her memory for both recent and past events was good. No evidence of hallucinations was elicited. At the time of admission she admitted having discontinued taking methimazole at least two weeks previously.

Results of complete blood cell count, sedimentation rate determination and urinalysis were within normal limits. Serologic tests for syphilis were nonreactive. The protein-bound iodine level on two determinations was 9.9 and 8.3 µgm per 100 ml and the serum butanol-extractable iodine level 7.8 and 6.4 µgm per 100 ml. Serum cholesterol was 207 mg per 100 ml. An electrocardiogram showed a sinus rhythm with a ventricular rate of 80 per minute.

No evidence of a substernal thyroid gland was seen in roentgenograms of the chest. X-ray films of the spine showed slight lumbosacral scoliosis, abnormal-appearing sacroiliac joints, gallstones and a stone in the left kidney. Repeated electromyograms showed some shortening of the anterior tibial muscles, but the records were interpreted as normal.

Because of the patient's lack of cooperation, the basal metabolic rate could not be determined. Results of other thyroid function studies performed at various intervals after cessation of antithyroid medication were as follows: radioactive iodine uptake (three weeks after the last dose of methimazole) was 40 per cent in one hour, 74 per cent in five hours, and 84 per cent in 24 hours. A triiodothyronine suppression test, with administration of 75 mg of triiodothyronine daily for three days and 100 mg daily for four days, was performed six weeks after the last dose of methimazole. Thyroidal uptake of radioactive iodine was 36 per cent in one hour, 64 per cent in five hours, and 63 per cent in 24 hours; uniform activity over both lobes was found by scan. Red-cell uptake of radioactive triiodothyronine (two weeks after the last dose of methimazole) was 19 per cent, and when repeated four weeks later was 23 and 20 per cent, respectively.

Chromatographic studies of the serum* showed increased amounts of triiodothyronine and thyroxin, but no abnormal iodinated compounds were found.

During a six-day period of observation, the patient received no medications other than sedatives at night. She remained depressed and extreme psychomotor slowing was still evident. On the seventh day treatment with an antidepressant drug, tranylcypromine, was begun. During the next week the initial daily dose of 30 mg was increased to 50 mg. This dose was maintained for two and one-half weeks, and the drug then was discontinued. The clinical status of the patient remained unchanged. The pulse rate ranged from 90 to 120 per minute, resting and waking. Her weight remained stable. In the fifth week of hospitalization electroconvulsive therapy was begun. The patient was given five treatments at intervals during the next two weeks. After the second treatment, her depression began to clear. After the fifth treatment the protein-bound iodine level on two occasions was 8.5 and 8.8 μgm per 100 ml, and radioactive iodine uptake was 42 per cent in three hours and 77 per cent in 24 hours. With the clearing of depression the patient suddenly developed clinical signs of thyrotoxicosis. She became hyperactive, and the pulse rate rose to 160 per minute. An electrocardiogram showed a sinus rhythm with a ventricular rate of 150. The patient's skin was warm and moist, and a hand tremor was present. The thyroid was enlarged; a bruit was audible. Treatment with chlorpromazine, 50 mg daily, and benztropine methanesulfonate, 2 mg daily, was ineffective in controlling the thyrotoxicosis and hyperactive state. The patient was then given a single dose of radioactive iodine, 4.2 microcuries, and during the next three weeks the hyperactivity gradually subsided. The therapeutic agents were then discontinued, and in their place chlordiazepoxide in doses up to 120 mg daily was substituted.

The patient was discharged on October 5, 1961, two and a half months after admission. She showed gradual improvement during the next five months and required fewer doses of sedatives. The protein-bound iodine level, determined one, two and five months after the patient's discharge from hospital, was 9.4, 4.4 and 7.7 μgm per 100 ml, respectively. Radioactive iodine uptake, determined at the same intervals, was respectively 3 per cent, 4 per cent and 13 per cent in five hours. When last seen five months after discharge, the patient was well adjusted and able to perform her household duties and community activities without difficulty.

*Chromatographic studies were carried out by a method as yet unpublished. Serum was obtained 24 and 48 hours after an oral dose of 100 microcuries of radioactive iodine and chromatographed on a Dowex 1x2 ion exchange resin, using a technique of gradient elution with increasing concentrations of acetic acid. Fractions containing iodoprotein, iodotyrosine, iodothyronine, and iodide were obtained, and radioactivity was determined.

DISCUSSION

At the time of admission to the hospital, the patient described in this report was in severe psychotic depression. Although the history and laboratory data were suggestive of hyperthyroidism, she showed little clinical evidence of hypermetabolic state. Also, her behavior, the depression and lack of agitation, as well as the persistence of pronounced psychomotor slowing, appeared inconsistent with such a diagnosis. The behavior of a hyperthyroid person is said to depend on his underlying personality[3,9] and the extent of the disease.[15] Even the occurrence of hyperthyroidism is thought by some observers to have a psychological as well as physiological basis. Investigators have commented on the incidence of the disease in persons who, like the patient here reported upon, are overly ambitious, moralistic, insecure and dependent, and have a compulsion to serve others through work.[2,10,15,16] In characteristic fashion, the patient in the present case was able to handle her psychic conflicts at first by working hard at home and in church activities. When hyperthyroidism associated with fatigability prevented control of anxiety in this manner, she became depressed, and the depression gradually became psychotic in depth. Such depression has been described as "a pathological state of conscious psychic suffering and guilt, accompanied by a marked reduction in the sense of personal values and diminution of the mental, psycho-motor and even organic activity, unrelated to actual deficiency."[18] In the patient herein reported upon, the depressive state had almost completely obscured the underlying hyperthyroid state. Although uncommon, other cases of hyperthyroidism masquerading as psychotic depression of sufficient severity to necessitate putting the patient in hospital have been reported.[3,12] In these patients the symptoms of psychosis, which were variable and in some instances associated with schizophrenic manifestations, disappeared after treatment of the hyperthyroidism by thyroidectomy.

Neither the patient in the present case nor three patients described in the literature[3,12] had symptoms resembling those of the central and autonomic hyperactivity characteristic of hyperthyroidism. Why these manifestations do not appear in the psychically depressed hyperthyroid patient is not known. Whether the increased metabolism in hyperthyroidism affects the brain tissue is debatable.[20,21] A number of studies referred to by Brewster[4] suggests that the level of thyroid hormone influences the sensitivity to the metabolic and cardiovascular actions of the catecholamines in both man and experimental animals. In his own experiments with dogs, Brewster showed that total sympathetic blockade induced by epidural anesthesia will reverse the increase in

oxygen consumption, in heart rate and in the force of ventricular contraction produced by thyroid hormone. Infusion of either epinephrine or norepinephrine will then result in a rise in these parameters, greater than that found in similarly treated euthyroid dogs. If this is true in man, it is possible that, in the patient in the present case, central suppression of the sympathetic nervous system may have prevented the usual manifestations of autonomic hyperactivity. Lending circumstantial support to this conjecture is the patient's clinical response to electroshock therapy. After the depression subsided, the usual symptoms of hyperthyroidism then developed, including those resembling the manifestations of excessive autonomic activity.

SUMMARY

In a patient with hyperthyroidism described in this report, a psychotic depression was associated with a decrease in the hypermetabolic and hemodynamic manifestations of the disease. After the depression had cleared in response to electroshock treatment, the typical signs and symptoms of hyperthyroidism developed. The clinical features and course in this case suggested that the hyperactivity and increased metabolism characteristically found in hyperthyroidism may have been suppressed by the psychotic depression.

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Lymphangioma of the Parotid Gland

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LYMPHANGIOMA of the parotid gland is an uncommon lesion. It is not mentioned in several comprehensive reviews[1,2,9] of parotid tumors and only occasional case reports are available.[4,8,11] This tumor characteristically occurs in infants and young children and is closely related to the more common lymphangioma of the neck (cystic hygroma colli) which is seen in the same age group. Owing to the rarity of primary parotid lymphangioma, the following case is presented.

REPORT OF A CASE

An 11-month-old white girl was admitted to San Francisco General Hospital on May 16, 1960, because of a mass over the angle of the left mandible. The tumor had been present since birth and had apparently increased in size only in proportion to the infant's somatic growth without noticeable fluctuation in its size or the development of obvious symptoms referable to its presence. Only an occasional mild infection in the upper respiratory tract was remarkable in the patient's medical history.

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